AIDS
THE SELENOENZYME SOLUTION

Eating foods grown in selenium-deficient soils or having a prior infection by a selenium-encoding pathogen are factors which promote susceptibility to HIV infection and ultimately AIDS.

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I don't try to describe the future. I try to prevent it.

– Ray Bradbury

THE MOST PROBABLE FUTURE

In 1992, in a lecture to the French Academy of Sciences, William A. Haseltine pointed out that "the future of AIDS is the future of humanity". Haseltine,[1] then the chief retrovirologist at Harvard's Dana-Farber Cancer Institute, went on to add that "Unless the epidemic of AIDS is controlled, there is no predictable future for our species". Later, testifying at a US Senate hearing, he predicted that by the year 2000 we could expect 50 million people to have been infected by HIV.[2] In his opinion, by 2015 the total number of dead or dying could reach one billion—that is, some sixth of the current global population.

Time has proven Haseltine to have been over-optimistic. By the end of 2000, an estimated 57.9 million people had been infected by HIV, 21.8 million of whom were already dead.[3] Current figures suggest a total of 70 million people have become HIV seropositive since the pandemic began in the early 1980s.[4]

As a consequence of our inability to halt the spread of HIV/AIDS, several of the worst-affected countries in sub-Saharan Africa are now on the verge of total social collapse as life expectancies, productivity, tax revenues and GDP dramatically fall and the need for expanded healthcare rises. There are many signs that suggest this situation will continue to worsen rapidly in the foreseeable future.[5]

PAST FAILURES

At an over-optimistic press conference held in 1984, Margaret Heckler, at that time the US Health and Human Services Secretary, announced the discovery of HIV, the virus believed responsible for the AIDS pandemic. She then went on to predict that a vaccine against this virus should be available within five years.[6] Heckler was clearly no Nostradamus, since almost 20 years later—after the expenditure of untold billions of research dollars—there is still no effective vaccine against either HIV-1 or HIV-2. Of course, there is no shortage of those willing to continue the expensive search to find one.

In June 2003, for example, 24 co-authors, including Nobel Prize winners, college presidents, heads of major US public health departments and AIDS researchers from around the world combined to argue for a Manhattan Project against AIDS. This, of course, would focus its efforts on the discovery of the long-awaited vaccine against HIV.[7] While there's no doubting the need for such a vaccine, there seems to me to be a very distinct possibility that it will not be available before 2015 and the infection of one sixth of the global population.

The news is not much better from the treatment front. HIV-1 exhibits at least two characteristics that make it extremely difficult to eradicate. Firstly, it lacks the ability to "proofread" its genetic sequences during replication.[8] The large number of resulting genetic errors results in the creation of endless variants, some of which inevitably will be immune to the antiretroviral drugs being used in treatment. As a consequence, inhibitors of reverse transcriptase and protease have promoted the evolution of drug-resistant strains of HIV that are now spreading rapidly in the developed world.[9, 10] At least one of these new strains is resistant to all three classes of drugs that are currently used to treat HIV/AIDS. Patients infected by this new strain have gone from being totally asymptomatic to having fully developed AIDS within a few months.[11] The treatment situation is

also made worse by the overdependence on AZT, a drug which is definitely carcinogenic.[12]

A second characteristic of HIV which makes infection by it so difficult to treat is the virus's ability to enter "resting" T-cells.[13] Such cells are particularly good places for a virus to hide because they are inactive and, therefore, ignored by the immune system. Similarly, "resting" T cells are not targeted by drugs, which in order to work also require some form of activity by either the infected cell or the virus. Since such "resting" T cells can remain dormant for years, even decades, HIV can exist undetected in infected individuals for a similar length of time.

HALTING THE AIDS PANDEMIC

Throughout recorded human history, pandemics have ravaged the known world. Typically, millions died from infection by a particular pathogen which then retreated, only to return later as community immunity declined. Cholera, influenza, typhoid, smallpox and bubonic plague, for example, have taken repeated heavy tolls of the human population.[14] There is, however, no convincing evidence of repetitive AIDS pandemics. The current scourge, already threatening to overtake the devastation associated with the Black Death, appears to be the first.

Simian immunodeficiency viruses (SIV) have been collected from 26 different species of African non-human primates. Two of these appear to have given rise to HIV-1 and HIV-2 in humans.[15] That is, these human viruses evolved from simian viruses as a result of zoonotic, cross-species transmission. A close examination of the genomes of these viruses seems to indicate that HIV-1 originated as the chimpanzee (*Pan troglodytes*) virus SIVcpz, while SIVsm, a sooty mangabey (*Cercocebus atys*) monkey virus, gave rise to HIV-2.

Numerous clinical trials have demonstrated that individuals eating a high-selenium diet are relatively unlikely to develop a wide variety of cancers.

However, mankind has been in close contact with chimpanzees, sooty mangabeys and other non-human primates for hundreds of thousands of years. Obviously there must have been endless opportunities through hunting and the bushmeat trade for human exposure to simian body fluids and for the cross-species transmission of viruses. Why, then, did HIV only begin to infect the human population on a global scale, for the first time, in the last two decades of the 20th century? After all, the 16th to 19th centuries saw the inhumanity of the slave trade, with the movement of millions of West Africans to Europe, North America and elsewhere. Had HIV-1 or HIV-2 been endemic in West Africa at the time, these viruses would certainly have been diffused around the globe by both slaves and slavers. Indeed, exotic diseases were spread by the slave trade from Africa to Europe and elsewhere; these included yellow fever, but they did not include AIDS.[16]

Viruses are like all other life-forms: they thrive in specific physical and social environments, and not in others. The most likely reason why HIV/AIDS is pandemic now is that certain changes in the environment, occurring in the latter part of the 20th and early part of the 21st century, have greatly improved HIV's competitive position.

What these changes were can be deduced from the work of E. W. Taylor and his colleagues at the University of Georgia. In the mid-1990s, these researchers discovered there was a series of viruses that encoded for a selenium-dependent glutathione peroxidase. These included HIV-1 and HIV-2, Coxsackievirus B, and the hepatitis B and C viruses.[17-19] What this means is that the genomes of such viruses include a gene that is virtually identical to that seen in humans, which allows them to manufacture the essential enzyme glutathione peroxidase. Subsequently, to prove that this apparent section of the HIV-1 genetic code really permitted it to produce the mammalian selenoenzyme glutathione peroxidase, Taylor and his co-workers[20] cloned the hypothetical HIV-1 gene and transfected canine kidney cells and MCF7 cells with it. In both cases, the cells given the HIV-1 gene greatly increased their production of the selenoprotein glutathione peroxidase. This proves beyond any reasonable doubt that HIV-1 (and probably HIV-2, Coxsackievirus B and the hepatitis B and C viruses) is capable of producing glutathione peroxidase for its own purposes.

More or less simultaneously, K. D. Aumann and co-workers,[21-23] of the Department of Biological Chemistry, University of Padova, Italy, were studying the biochemistry of the glutathione peroxidases. In three articles, they argued that glutathione peroxidase is characterised by catalytically active selenium which forms the centre of a strictly conserved triad composed of selenocysteine, glutamine and tryptophan. That is, they believed that it consisted of the trace element selenium and three amino acids, namely cysteine, glutamine and tryptophan. Their suggestion, it should be noted, ran contrary to the conventional belief that glutathione peroxidase consists of selenium, cysteine, glutamine and not tryptophan but glycine.

Regardless of the true composition of glutathione peroxidase, there is no doubt that this enzyme contains selenium. Since, as researchers at the University of Georgia have established, HIV-1 and HIV-2, Coxsackievirus B and the hepatitis B and C viruses all encode for this enzyme, it would seem logical to expect that infections from them would peak in high-selenium regions. Interestingly, there is abundant evidence that the reverse is true and that a high dietary selenium intake gives a great deal of immunity against all of these viruses.[24]

Indeed, it is believed by the author that this inability to diffuse, in areas where the population has a relatively high selenium intake, represents the Achilles heel of HIV/AIDS and currently offers the best available strategy for halting, or at least slowing, the pandemic.[25]

In sub-Saharan Africa, Senegal stands out like a diamond in the dirt. Given the widespread polygamy and unprotected promiscuity in the country,[26-27] one would expect that its mortality from AIDS would have been enormous. After all, Senegal is located in sub-Saharan Africa, close to the region where the simian immunodeficiency virus (SIVcpz) is believed to have been transmitted from chimpanzees to humans on several occasions and where it subsequently evolved into HIV-1. However, in Dakar, Senegal's major urban centre, HIV-1 prevalence among women attending antenatal clinics has remained at one per cent or less since the time that surveillance began in the mid-1980s until the present. Similar very-low-prevalence rates are also recorded in the Senegalese hinterland.[28]

Geologically, Senegal is a dried-up Cretaceous and early Eocene sea. When this dessication took place, sedimentary rocks were formed from the dissolved minerals in evaporating sea

water. As a result, calcium phosphates now mined for use in fertilisers are one of Senegal's chief mineral products. They are derived from phosphorite, a rock type that is always selenium-enriched.[29]

It appears to be no coincidence that HIV-1 has had great difficulty diffusing in Senegal, a country which also has the world's lowest incidence of cancer.[30] Numerous clinical trials, of course, have demonstrated that individuals eating a high-selenium diet are relatively unlikely to develop a wide variety of cancers.[31]

Conversely, a link between elevated AIDS mortality and depressed environmental selenium has been shown to occur in the United States. Cowgill,[32] for example, used analysis of variance to compare selenium in local alfalfa with AIDS mortality for 1990. Where selenium levels were depressed, AIDS mortality was elevated. This relationship was particularly evident amongst Afro-Americans, who Cowgill believed were less mobile and therefore more likely to eat locally grown foods. This inverse relationship between dietary selenium intake and risk of infection does not seem limited to HIV-1, but also appears to be true of other viruses that encode for glutathione peroxidase.

Beyond that, Beck and her co-workers,[33] for example, have shown that a normally benign Coxsackievirus can mutate to cause significant heart damage in selenium-deficient mice. Such new viral strains differed significantly from the original virus and were also then able to cause heart problems in selenium-adequate animals.

This relationship between the virulence of the Coxsackievirus and heart disease in mice is of more than just academic concern. A frequently fatal cardiomyopathy called Keshan disease is widespread and endemic in the selenium-deficient areas of China.[34] It occurs in those who are both selenium deficient and infected by the Coxsackievirus. It is therefore a disease caused by a virus that encodes for glutathione peroxidase, but only infects those who are eating a diet containing inadequate selenium.

This problem may not be limited only to regions of extreme selenium-deficiency. Nicholls and Thomas,[35] for example, showed that 10 out of 38 patients suffering acute myocardial infarction (heart attack), admitted to the King Edward VII Hospital in Midhurst, Sussex, England, during a two-month period, had serological evidence of very recent Coxsackievirus B infection. That is, approximately 25 per cent of these British heart attack patients had suffered from an influenza-like illness caused by the Coxsackievirus B within seven days prior to admission. Even more interesting is the fact that heart attack patients who subsequently took selenium supplements suffered far fewer secondary episodes of myocardial infarction.[36, 37]

Further evidence that selenium supplementation can greatly reduce infection by the Coxsackievirus has been provided from China, where the incidence and mortality rates for Keshan disease are in decline.[38] This is because of the widespread use of more grain grown outside the selenium deficiency belt, spraying selenium-enriched fertilisers onto soils and crops, and adding this trace element to the feed of domestic livestock and to table salt. To illustrate, in Sichuan Province[39] the use of selenium-fortified table salt was able to reduce the incidence of Keshan disease in children from 7.1 to 0.12 per thousand during the period 1974 to 1983. Everywhere in the great Chinese selenium deficiency belt, as the level of this trace element has risen in local diets Coxsackievirus infection has fallen and, with it, Keshan disease incidence and mortality.[40]

The Chinese also have provided evidence that increased dietary selenium can reduce the rates of infection by two more pathogens that encode for glutathione peroxidase: the hepatitis B and C viruses. In Qidong County, Jiangsu Province,[41] 20,847 residents of one town were given table salt fortified with 15 ppm of anhydrous sodium selenite. Those in the six surrounding townships continued to use normal table salt. Prior to and during the first year of the study, there was no statistically significant difference in hepatitis infection between the selenium supplementation and control populations. However, by the third year, a drop in the incidence of hepatitis had occurred in the selenium-supplied township (4.52 per 1,000) compared with those communities using normal salt (10.48 per 1,000; 56.8% reduction, $p<0.002$). A similar study in the same county, also conducted by Yu and colleagues,[42] further established that daily selenium-yeast (200 micrograms of selenium) supplementation could significantly reduce the primary liver cancer often associated with hepatitis B and C infection. Interestingly, Berkson[43] has demonstrated that the liver damage caused by hepatitis C can be reversed by a combination of alpha-lipoic acid, silymarin and selenium, often negating the need for expensive liver transplantation.

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In summary, infection from HIV-1, Coxsackievirus B and the hepatitis B and C viruses occurs far more frequently in regions and populations that are selenium deficient. It has been established further that rates of infection by and death from Coxsackievirus B and hepatitis B and C viruses can be greatly reduced by increasing dietary selenium intake. It seems extremely likely, therefore, that the same strategy would be just as effective in slowing the diffusion of HIV-1 and so lowering the AIDS death rate.

Unfortunately, the reverse seems to be occurring. During the latter half of the 20th century, precipitation became increasingly acidic, soil pH fell, and heavy metal and fertiliser contamination increased. As a consequence, selenium bioavailability declined and levels of this element in the food chain fell,[44] making it much easier for viruses that encode for glutathione peroxidase to diffuse. This is why we are now experiencing pandemics caused by HIV-1, the Coxsackievirus and the hepatitis B and C viruses.[45, 46] Together they have infected more than one third of the global human population and show no sign of halting their rapid spread. Their devastation, of course, is most obvious in those regions of the planet where, for geological reasons, the soil levels of selenium are naturally very low. These include most of sub-Saharan Africa and the "disease belt" that crosses China from northeast to southwest.

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If we are going to have any hope of halting the AIDS pandemic and of slowing the diffusion of hepatitis B and C, the dietary intake of selenium must be increased in such areas. It is clear also that, even in the developed world, additional selenium could greatly reduce cancer incidence and lower mortality from myocardial infarction.[47, 48]

THE REVERSAL OF AIDS

After infection with HIV-1 there is an initial brief illness, with lymph node enlargement and fatigue. These symptoms are like those of mononucleosis, but far more transient. However, usually several years later, diverse new symptoms occur that typically include night sweats, diarrhoea, psoriasis, muscle wasting, immune incompetence and depression.[49] In Africa, it appears to take some five years after initial infection until the development of AIDS, which is characterised by these symptoms. In the developed world, this period is somewhat longer, probably nearer 10 years.[50]

Many and varied hypotheses have been put forward to explain how HIV-1 causes AIDS.[51] Unfortunately, they appear unconvincing since they tend to focus on immune incompetence and do not adequately explain the wide range of other symptoms seen in AIDS patients, including the abnormal incidence of Kaposi's sarcoma.

Recently in my book, *What Really Causes AIDS*,[52] I put forward an alternative hypothesis that not only explains why HIV-1 takes so long to cause AIDS but why this disease has the specific symptoms it does. It was suggested that since HIV-1 encodes for the human selenoenzyme glutathione peroxidase, as it is replicated its genetic needs cause it to deprive seropositive individuals not only of glutathione peroxidase but also of its four basic components: selenium, cysteine, glutamine and tryptophan. Eventually, after a period of time (the length of which depends on the diet being eaten), this depletion process causes severe deficiencies of all these nutrients.

In Africa, it appears to take some five years after initial infection until the development of AIDS… In the developed world, this period is somewhat longer, probably nearer 10 years.

These in turn are responsible for the major symptoms of AIDS, which include immune system collapse, increased cancer and myocardial infarction susceptibility, muscle wasting, depression, psychosis, dementia and diarrhoea. Naturally, since these nutritional deficiencies cause immune system failure, other pathogens can infect the patient and become responsible for their own unique symptoms.

One of these symptoms is Kaposi's sarcoma, which is linked to the human herpes virus 8 (HHV-8), a virus that was endemic for years in Uganda and other selenium-deficient regions of sub-Saharan Africa long before the onset of AIDS.[53]

If this hypothesis is correct, four corollaries must follow.

• Firstly, AIDS patients should be very deficient in glutathione peroxidase and its components selenium, cysteine, glutamine and tryptophan.

• Secondly, any effective treatment for HIV/AIDS must include normalisation of body levels of glutathione, glutathione peroxidase, selenium, cysteine, glutamine and tryptophan.

• Thirdly, since deficiencies of these nutrients cause the main symptoms of AIDS, correcting them should reverse the disorder. The only symptoms remaining might be expected to be those caused by other opportunistic pathogens.

• Fourthly, since the symptoms of AIDS are those of extreme deficiencies of one trace element and three amino acids, it follows that individuals who are HIV-1 seropositive but who eat diets elevated in these four nutrients should never develop AIDS.

Evidence exploring these four corollaries is presented in part two of this series.

Continued next issue...

Author's Note:

Readers wanting more detailed information about the HIV/AIDS environmental link are directed to the website http://www.hdfoster.com, where they can download a free copy of my book, *What Really Causes AIDS*.

About the Author:

Harold D. Foster, PhD, was born and educated in England. He specialised in geology and geography, earning a BSc in 1964 from University College London and a PhD in 1968 from London University. He is a Canadian by choice, and has been a faculty member in the Department of Geography, University of Victoria, British Columbia, Canada, since 1967.

A tenured professor, Dr Foster has authored or edited some 235 publications, the majority of which focus on reducing disaster losses or identifying the causes of chronic disease or longevity.

He has published hypotheses on the origins of numerous diseases including myocardial infarction, SIDS, cancer, diabetes, schizophrenia, multiple sclerosis (MS), amyotrophic lateral sclerosis (ALS), Alzheimer's and Parkinson's diseases, and stroke.

His numerous books include: *Disaster Planning: The Preservation of Life and Property* (Springer Verlag, New York, 1980); *Reducing Cancer Mortality: A Geographical Perspective* (Western Geographical Press, Victoria, 1986); *The Ozymandias Principles: Thirty-one Strategies for Surviving Change* (Southdowne Press, Victoria, 1997); and *What Really Causes AIDS* (Trafford Publishing, Victoria, 2002; see review in *NEXUS* 10/05). His new book, *What Really Causes Schizophrenia*, is to be published by Trafford in late 2003.

Harold Foster is a member of the Explorers Club as well as several academic organisations including The New York Academy of Sciences, The Royal Geographical Society and The Royal Society of Literature. He is also the editor of both the International and Canadian *Western Geographical Series* and is a member of the boards of the *Journal of Orthomolecular Medicine* and the International Schizophrenia Foundation.

He has been a consultant to numerous organisations, including the United Nations and NATO, and to the governments of Canada, Ontario and British Columbia. He is also a member of the Science Advisory Panel for the Healthy Water Association.

Every day, Dr Foster makes a point of taking at least the recommended daily allowance of the known essential nutrients. He is also currently pursuing offers for his suggested nutrient mixture to be produced for use in clinical trials with AIDS patients. For a more detailed résumé, visit the website http://www.hdfoster.com.